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Research Interests

Our laboratory is focused on elucidating the molecular mechanisms responsible for cardiac muscle injury in diabetes. We are examining the role of altered insulin signaling and altered glucose utilization. To model this, we have generated mice with cardiomyocyte-restricted deletion of the insulin receptor and other molecules in the insulin signaling pathway (such as PI3 kinase and GLUT4). We have used these models to define the link between altered insulin signaling and the onset of mitochondrial dysfunction and impaired cardiac substrate metabolism that characterizes the heart in diabetes. We have taken a broad approach that includes *in vivo* assessment of cardiac function in mice; analyses of isolated hearts, isolated cardiomyocytes, and isolated mitochondria; and gene expression profiling.

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